ORIGINAL COMMUNICATIONS

STUDIES ON LEAD EXPOSURE IN PATIENTS OF A NEIGHBORHOOD HEALTH CENTER: PART II. A COMPARISON OF WOMEN OF CHILDBEARING AGE AND CHILDREN

George D. Flanigan, Jr, MD, Robert Mayfield, MT, ASCP, and Herman T. Blumenthal, PhD, MD
St Louis, Missouri

The second part of this study deals with blood lead (PbB) levels in 541 pregnant women and 351 nonpregnant women of childbearing age residing in the health center's catchment area. Each blood sample was analyzed for erythroprotoporphyrin (EP) and PbB. Comparisons of these data were made with national data on women of childbearing age and with children in our catchment area, as well as with children nationally.

Mean PbB and prevalence rates at ≥10 and ≥15 μg/dL were higher in the nonpregnant women of the catchment area than in the pregnant women. For comparison with women nationally, the data on these two groups were combined because national data did not clearly distinguish between pregnant and nonpregnant women.

The women in the catchment area showed mean PbB and prevalence rates at the two ranges noted above that were not only higher than in white women but also generally higher than in black women nationally. Thus, this rank order was similar to that reported earlier for children.[1]

The comparisons between women and children living in the same environment showed that the children have significantly higher mean PbBs and prevalence rates at the two ranges than the women. The physiological and environmental bases for these differences are discussed.

As in the study on children,[1] the EP is also not adequate as a screening procedure for identifying women with PbB levels ≥25 and ≥15 μg/dL. (*J Natl Med Assoc.* 1992;84:23-27.)

Key words • blood lead levels • childbearing-age women • erythroprotoporphyrin

From the Department of Community Medicine, St Louis University School of Medicine and the St Louis Comprehensive Neighborhood Health Center, St Louis, Missouri. Requests for reprints should be addressed to Dr Herman T. Blumenthal, Dept of Community Medicine, St Louis University School of Medicine, 1320 S Grand Blvd, St Louis, MO 63104.

As reported in Part I,[1] children between the ages of 6 months and 5 years who resided in the catchment area of the St Louis Comprehensive Neighborhood Health Center (SLCNHC) had, for more than a decade, exhibited exceptionally high mean blood lead (PbB) levels as well as high prevalence rates at toxic levels. It was also noted that reports[2-11] showed that lead crosses the placental barrier during pregnancy and that pregnant women with PbB levels as low as 10 to 15 μg/dL may give birth to infants with behavioral and neurological deficits that are not reversed by the end of the first year. In the current study, we attempt to determine the extent to which maternal PbB levels reflect the levels in

TABLE 1. MEAN PbB LEVELS AND PREVALENCE RATES AT SPECIFIED RANGES, NATIONAL VERSUS SLCNHC

Population	N	Age Group	Mean PbB (μg/dL)	Prevalence (%) <10	>10	>15
National 1984 estimates*		15-44 yrs				
White women	41.3 million		4.6	90.5	9.5	1.4
Black women	3.6 million		6.9	82.4	17.6	3.3
SLCNHC 1988-1989		15-44 yrs				
Pregnant women	541		7.9	73.2	26.8	5.7
Nonpregnant women	315		8.8	65.5	34.5	12.1
Combined	856		8.2	70.2	29.8	8.1
National 1984 estimates†		0.5-5 yrs				
White children	41.3 million		—†	—	—†	13.2
Black children	3.6 million		—	—	—	40.3
SLCNHC 1989		0.5-5 yrs				
Children	208		15.1	25.0	75.0	39.9

*Data were derived from Tables VII-1 and VII-2 of the ATSDR Report. Table VII-2 presents the number of women in the base population and at each of the PbB ranges. It also divides the data into separate categories of "Women of Childbearing Age" and "Pregnant Women." When the prevalence rate at each PbB range was calculated the two categories showed identical prevalence rates. These were the same as in Table VII-1, which has only the single designation of "Women of Childbearing Age."
†Data were derived from Table V-3 of the ASTDR Report, which does not provide mean or >10 data.

children in the catchment area. The sensitivity and specificity of the erythroprotoporphyrin (EP) method was examined again to determine if this screening procedure is more reliable in adults than in children.

METHODS

Blood samples for EP and PbB determinations were routinely obtained between 1988 and 1989 from 856 black women. Of these women, 541 were in the first trimester of pregnancy and 315 were nonpregnant women of childbearing age. The data from these analyses were compared with those in white and black women in the 1984 Agency for Toxic Substances and Disease Registry (ATSDR) report,[12] the most recent year for which national data are available. For comparison of PbB parameters in women and children, the data on children for the year 1984 nationally and the year 1989 for the catchment area have been extracted from our first report.[1]

Erythroprotoporphyrin and PbB determinations were carried out as described in the preceding report.[1] Mean PbB levels and prevalence rates at ranges $\geqslant$10 and $\geqslant$15 μg/dL were calculated from the results of these analyses. The sensitivity and specificity of the EP as a screening procedure in adult women also were determined as in the preceding report.[1]

RESULTS

Comparison of PbB Parameters in SLCNHC Women and Women Nationally

Table 1 compares national 1984 estimates of PbB parameters in women of childbearing age with definitive 1989 data on pregnant women and women of childbearing age attending the SLCNHC. The national data do not clearly distinguish between pregnant and nonpregnant women, as explained in the footnote to Table 1. In the SLCNHC population, nonpregnant women showed a higher mean PbB level than pregnant women, as well as higher prevalence rates at ranges $\geqslant$10 and $\geqslant$15 μg/dL. However, for comparison with national data, the two groups of women were combined.

The mean PbB level in all of the groups shown in Table 1 are under the 10 μg/dL level. Nevertheless, the mean PbB level in black women nationally is 1.5 times higher than in white women. In the SLCNHC population, the combined data on pregnant and nonpregnant women showed a mean PbB level almost twice as high as in white women and about 19% higher than in black women nationally.

Table 1 also shows that more than 90% of white women of childbearing age have PbB levels below 10 μg/dL compared with more than 80% in black women nationally and only 70.2% of the women in the catchment area. As to prevalence rates ≥10 μg/dL, black women nationally show a rate almost twice as high as in white women, while the women in the catchment area show a prevalence rate more than three times that found in white women and more than 1.5 times that found in black women nationally.

While prevalence rates at a range ≥15 are low in all three groups of women, the rate in black women nationally is almost 2.5 times that in white women. The women in the catchment area show a rate almost six times that found in white women and about 2.5 times that found in black women nationally. Twenty-one of the women in the SLCNHC population (2.5%) showed PbB levels ≥20, with the highest level being 39 μg/dL. Thus, if the criterion of ≥10 μg/dL is considered to place the fetus at risk for brain damage, then almost one third of the pregnancies in the SLCNHC population place the fetus at such risk. If the criterion of ≥15 μg/dL is used, more than 8% of the pregnancies in the SLCNHC population are at such risk.

Comparison of PbB Parameters in SLCNHC Children With Children Nationally

The data in the lower portion of Table 1 reflect the PbB parameters in children and are largely a reiteration of data presented previously.[1] They are presented here for the convenience of comparing the data in children with those in adult women.

National 1984 estimates do not provide mean PbB levels or prevalence rates below and over 10 μg/dL. As noted previously,[1] the data for the SLCNHC population show a significant decline in mean PbB levels between 1984 and 1989. As shown in the lower portion of Table 1, black children nationally in 1984 and those in our population in 1989 showed a prevalence rate at a range ≥15 μg/dL about three times more often than in white children nationally. Also evident is the fact that PbB parameters are much higher in children both nationally and in the catchment area than in women.

Evaluation of EP Screening

Table 2 compares the sensitivity and specificity of EP between women and children at particular criteria. Both criteria show the sensitivity is higher in children than in women, but in both groups sensitivity is far below a desirable level. The comparisons with respect to specificity show a fairly acceptable specificity at the criterion PbB ≥25 and EP ≥35, but if the criterion is set at PbB ≥15 and EP ≥25, it is far below an acceptable level.

TABLE 2. SENSITIVITY AND SPECIFICITY OF EP IN WOMEN OF CHILDBEARING AGE: COMPARISON WITH CHILDREN

	Women	Children
Sensitivity		
Criterion: PbB > 25 spec by EP > 35	20.0%	39.4%
Criterion: PbB > 15 spec by EP > 25	45.2%	54.2%
Specificity		
Criterion: PbB > 25 and EP > 35	84.4%	84.3%
Criterion: PbB > 15 and EP > 25	55.9%	47.8%

To determine the extent to which anemia (which is known to produce false high EP values in lead screening) might account for these findings, the prevalence of subjects with hemoglobin levels below 11 g was determined. The actual range turned out to be 7.1 to 10.9. In the SLCNHC population, 24.6% of the women fell within this range. Thus, a fairly high prevalence of anemia was present as a confounding factor.

DISCUSSION

This study on the women in our catchment area was carried out between 1988 and 1989 after the older data on the children in the catchment area were discovered, as described in our first report.[1] The 1989 data on SLCNHC children were therefore selected for an appropriate comparison. However, the most recent available nationwide estimates on both women and children were from 1984. The data in the previous report[1] show that if the 1984 data on the SLCNHC children are compared with those nationally, then the differences in PbB parameters are greater than those shown in Table 1. If it is assumed, as appears likely for reasons noted in the previous report, that these PbB parameters in the SLCNHC women were also higher in 1984 than in 1989 or that women nationally had lower PbB parameters in 1989 than in 1984, then the differences between the SLCNHC women and those nationally would also be greater than those shown in Table 1.

Nevertheless, there are several generalizations that even these uneven comparisons permit. Blood lead parameters are much higher in children than in women of childbearing age both nationally and in the SLCNHC population, and in both women and children, blood lead levels are higher in blacks than in whites.

Even though mean PbB levels in women of childbearing age are <10 μg/dL, there is reason for concern if 10 to 15 μg/dL represents a level at which the fetus is at risk for brain damage. In the catchment area, only about 70% of the women had PbB levels under 10 μg/dL compared with about 90% in white and about 82% in black women nationally. Thus, at a range ≥10 μg/dL, less than 10% of white women and more than 17% of black women nationally fall within this range, while in the SLCNHC population the prevalence rate is almost 30%. At the range ≥15 μg/dL, white women nationally showed a prevalence rate of only 1.4% and black women 3.3%, while the SLCNHC women showed a rate of 8.1%—more than twice the rate in black women and about six times the rate in white women nationally.

The great difference in PbB parameters between children and women of childbearing age living in the same environment has several explanations. A Boston study[13] has shown that although as children grow older there is a decline in the number screened, the percent with PbB levels ≥25 or EP levels ≥35 remains about the same. Moreover, 58% of the children after age 2 with levels above these criteria had levels below these criteria at age 12 to 18 months, thus demonstrating additional retention of lead in their bodies as they grew older. If we had been able to divide our data on children into a 6 months-to-2-year and a 2-to-5-year group, the older group might have shown an even greater difference when compared with adult women. The ATSDR report[12] provides physiological explanations as to possible mechanisms that may explain the differences in PbB parameters between women and children:

1. Young children absorb larger amounts of lead per unit body weight than adults.
2. Children also retain a larger fraction of absorbed lead than do adults.
3. Children are not as efficient as adults at sequestering lead in their bones; a higher fraction of the total body lead burden is therefore available to exert toxic effects.
4. Basal metabolic and respiration rates are higher in children than in adults, and these enhance lead uptake and toxicity risks.
5. As noted previously,[1] nutrient status affects lead absorption and retention. Deficiencies in iron, calcium, and other elements commonly encountered in children living in poverty enhance lead uptake and retention and increase toxicity risks.

A recent study[14] on 34 adults (age range: 20 to 60 years), using in vivo x-ray fluorescence measurements of lead concentration in bones, showed that 16 had levels above 6 μg/g, defined as the level below which there is "measurement uncertainty." There was a positive correlation between bone concentration and age. These 16 had all lived in pre-1955 housing with lead-paint contaminated dust and lead-pipe contaminated water. This study confirmed previous reports on lead concentrations in cadaver bones. Thus, while only children may eat lead-contaminated paint chips and play in lead-contaminated soil, adults continue to have a significant exposure to lead and a progressive accumulation in bones. When women living in such an environment become pregnant, the lead in their bones becomes a source for lead in their blood and, in turn, the blood becomes the source of fetal exposure. Furthermore, fetal exposure to maternal lead represents only the beginning of a harmful process that progresses in children as the maternal source of exposure is replaced by environmental sources.

Finally, the reasons for resorting to EP as a screening procedure are not applicable to adults because there is usually no problem with venipuncture in adults. The EP procedure is cheaper, however, than definitive PbB determinations and may therefore be used in some screening programs. Nevertheless, the evaluation of EP sensitivity and specificity in adults has some value in that it provides an opportunity for assessing the impact of anemia on falsely elevated EP levels for lead screening purposes. Our rank order with respect to hemoglobin levels <11 g/dL is the same as with lead parameters. National data[15] show that only 3.5% of white women during the first trimester of pregnancy have anemia by this criterion, compared with 12.7% in black women nationally, while SLCNHC women show a rate of 24.6%. At any rate, it is evident that EP lacks both sensitivity and specificity in adults as well as in children for use in screening programs to detect either adults or children with PbB levels above the currently recommended 10 to 15 μg/dL.

Literature Cited

1. Blumenthal HT, Flanigan GD, Mayfield R. Studies on lead exposure in patients of a neighborhood health center, I: pediatric patients. *J Natl Med Assoc.* 1991;83:1065-1072.

2. Rabinowitz MB, Needleman HL. Temporal trends in the lead concentration of umbilical cord blood. *Science.* 1982;216:1429-1431.

3. Alexander PW, Delves HT. Blood lead levels during pregnancy. *Int Arch Occup Environ Health.* 1981;48:35-39.

4. Barltrop D. Transfer of lead to the human fetus. In: Barltrop D, Burland WL, eds. *Mineral Metabolism in Pediatrics. Proceedings of a Symposium Held in Greensford in 1968.* Philadelphia, Pa: FA Davis Co; 1969:135-151.

5. Ernhard CD, Wolf AW, Sokal HJ, Brittenham GM, Erhard P. Fetal lead exposure. Antenatal factors. *Environ Res.* 1985;38:54-66.

6. Bellinger D, Leviton A, Rabinowitz M, Needleman H, Waternaux C. Correlates of low level lead exposure in urban children under 2 years of age. *Pediatrics.* 1986;77:826-833.

7. Deitrich KN, Krafft KM, Bornschein HL, Hammond PB, Berger O, Succop PA, et al. Low-level lead fetal exposure on neurobehavioral development in early infancy. *Pediatrics.* 1987;80:721-730.

8. Bellinger D, Leviton A, Waternaux C, Needleman H, Rabinowitz M. Longitudinal analyses of prenatal and postnatal lead exposure and early cognitive development. *N Engl J Med.* 1987;316:1037-1043.

9. McMichael AJ, Baghurst PA, Wigg NR, Vimpani GV, Robertson EF, Roberts RJ. Port Pirie cohort study: environmental exposure to lead and children's abilities at age 4 years. *N Engl J Med.* 1988;319:468-475.

10. Wigg NR, Vispani GN, McMichael AK, Baghurst PA, Robertson EF, Roberts RJ. Port Pirie cohort study: Childhood blood lead and neuropsychological development at age 2 years. *J Epidemiol Community Health.* 1988;42:213-219.

11. Benetou-Marantidou A, Nakau S, Michelogannis J. Neurobehavioral estimation of children with life-long increased lead exposure. *Arch Environ Health.* 1988;32:392-395.

12. Agency for Toxic Substances and Disease Registry. *The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress.* Washington, DC: Government Printing Office; 1988. US Dept of Health and Human Services publication DHSS 99-2966.

13. Brown MJ, DeGiacomo JM, Gallagher G, Graef J, Leff J, Mathieu O, et al. Lead poisoning in children of different ages. *N Engl J Med.* 1990;323:135-136.

14. Hu H, Milder FL, Burger DE. X-ray fluorescence measurements of lead burden in subjects with low level community lead exposure. *Arch Environ Health.* 1990;45:335-341.

15. Centers for Disease Control. Anemia during pregnancy in low-income women—United States, 1887. *MMWR.* 1990;39(5):73-76.